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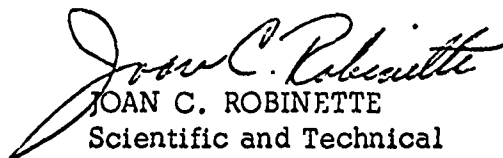ATTN OF AMRL/SCI   22 July 1970

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Biomechanics of the Vertebral Column and Internal Organ Response to Seated Spinal Impact in the Rhesus Monkey (Macaca Mulatta)[1]

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Abstract

Rhesus monkeys were anesthetized; radiographed; restrained by lap belt, torso harness, and limb retention straps; positioned in an impact carriage; and exposed to $+G_z$ seated rectangular acceleration-time histories from predetermined drop heights. The shock programmer used throughout these experiments was aluminum honeycomb. Pulse accelerations ranged 25–900 G and total time duration 2–22 ms. Shortly after impact, all primates were radiographed, killed, and a necropsy performed. Attempts were made to determine injury potential as a function of plateau acceleration and pulse duration for various target organs. Spinal injury data support previous results and theoretical considerations that there are two distinct injury potential regimes; one, for which the velocity change is the determining physical parameter, and the other for which peak g is the determining factor. Injuries observed in parenchymatous organs included external and surface hemorrhaging in the liver, lesions of the lung, and cardiac lesions. Their severity ranged from minor reversible abrasions to lethal trauma. These experiments strengthened confidence in the applicability of animal experiments to the quantitative explanation of human injury and to the extrapolation of animal results — after the application of the proper scaling factors — to human situations.

THE PROBLEM OF PREDICTING whether or not a particular acceleration input to a vehicle will result in trauma to its occupant has been studied in detail by a number of investigators using vertical towers (1–3)[3] horizontal sleds (4–8), vibration tables (9–16), and the human centrifuge (17). The results of these experiments have shown performance decrement and subjective endpoints to be related to the severity of mechanical loading. The "tolerance" curves for these various types of mechanical stress environments are usually presented as extremely broad bands of subjective tolerance, risk probability, or injury limit. Also, no indication is given as to which internal organ system proved to be the anatomically or physiologically most susceptible component within the torso to stimulate the particular "tolerance" response. Since it is unethical to expose human subjects to injurious environments, a factor of uncertainty exists in all those data about the actual acceleration-time history where frank structural injury will occur.

In addition to human experimentation, a number of investigators (21–25) have contributed significantly to an understanding of the mechanical and physical response characteristics of isolated hard and soft tissue taken from animals during necropsy and from human cadavers. German investigators were the first to subject systematically fresh motor segments of the human vertebral column to known compression loads and determine the force necessary to cause damage. The purpose of these tests was to develop meaningful acceleration/deceleration standards and minimize the vertebral fractures observed in airmen following belly landings or landing on skids in German aircraft. Although soft tissue trauma was also present, there were not enough cases to provide adequate statistical significance.

The experimental findings of Ruff (26), concerning vertebral body breaking strength, agreed with the earlier findings of Wischofer (27) and Richter (28), and led to the first tolerance data for abrupt longitudinal acceleration. The results of these experiments demonstrated that the human body could withstand input accelerations of up to 20 G. Meanwhile, Geertz (29, 30) studied the biodynamics of abrupt accelerations through multi-degree mechanical/mathematical analogs, established acceleration plateaus for headward acceleration, and noted that the dynamic response of a biologic system could be greater than the peak acceleration of the input pulse

The results of those experiments — combined with the experimental data based on human volunteers, cadavers, and isolated tissue — have led to increased interest in theoretical studies to develop mathematical ap-

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[3]Numbers in parentheses designate References at end of paper.

proaches as aids in solving the problems of human tolerance to impact in present day situations.

Simple analytic mechanical models of the human torso are used as a basis for a theory to correlate and explain current experimental observations with human subjects. The process of accumulating experimental data and refining the mathematical models is repeated and extended until the particular mathematical analogy produces results similar to the response observed on human subjects in various acceleration-time histories.

Model techniques have been particularly successful in explaining the effects of buttock-to-head acceleration. Vertebral body damage is usually assumed to be the primary mode of injury — the one occuring at the lowest exposure level. Although additional modes of injury to the various internal organs are known to occur, quantitative information on internal organ dynamics and injury probability, along with clinical dysfunction and symptomatology, is almost nonexistent. Consequently, the mechanisms of injury to the various components within the torso are not understood in enough detail to be quantitatively compared with the injury predictions resulting from the use of higher degree of freedom mathematical models (31–35).

For the above reasons it is desirable to have well-defined structural tolerance levels for those organs most susceptible to injury for a particular environment. However, because of the multiple variables encountered in both man (age, anthropometric characteristics) and the dynamic environment (the range of mechanical loading to which he may be subjected is quite wide, both in terms of frequency, direction, and amplitude), definition of these limits is difficult. However, tolerance limits can be established for a particular body axis.

It is the purpose of the present study to identify, with more quantitative precision than has previously been possible, injury limits for various organs and tissues as a function of the physical stimulus parameters. One of the goals of this series of experiments is the establishment of curves of equal injury probability for various organs, as a function of acceleration magnitude and duration. Rectangular acceleration pulses were selected because of their well-defined characteristics and their practical as well as theoretical significance and interpretability. The acceleration inputs were presented as $+G_z$ spinal accelerations, because the injury mechanisms for this directional loading are best understood, and the results promised to be more easily explained and incorporated into existing theoretical injury models. Although the increased complexity of body dynamics for impact inputs from other directions is realized, it is hoped, nevertheless, that the basic knowledge and methodology evolving from the $+G_z$ studies can be applied in subsequent studies involving the other body axes. In order to evaluate the importance of symmetrical loading of the

spinal column on the probability of vertebral injury and the levels of soft tissue trauma, G_z exposures were conducted with the spinal column aligned for optimum load bearing (Experiment 1), and with a curved spine resulting in asymmetrical anterior vertebral body loads (Experiment 2).

The study was conducted with Rhesus monkeys. It is one of a series of experiments to expose systematically subhuman primates within the primate hierarchy to controlled G-time histories. It will also establish the experimental basis for more quantitative scaling laws supporting theoretical efforts aimed toward developing the capability of extrapolating human impact tolerance levels from animal experiments.

This paper describes the bony alterations and soft tissue trauma, as well as a surface wave phenomenon that we observed. The detailed pathological findings, correlation of the Rhesus monkey data with the results of studies on species of different size as well as application of the data to the refinement of model calculations, and scaling laws must be left to future reports.

Experimental Equipment

To accomplish this study, the vertical deceleration tower at the Aerospace Medical Research Laboratory was modified in order to produce the desired acceleration-time histories. Two vertical, ½ in. diameter steel cables, aligned parallel to each other 36 in. apart and pretensioned to 1400 lb, served as low friction guide cables along which the impact vehicle and payload (guided by four Teflon-lined eyebolts rigidly anchored into its sides) was raised to a predetermined drop height, released from rest, allowed to accelerate, and impacted onto a honeycomb decelerator. The impact vehicle was constructed of glued and doweled laminated maple wood. Its corners were reinforced with welded ½ in. steel plates. The support system, visible within the shell of the vehicle (Fig. 1), was constructed of ⅜ in. mechanically coupled and welded aluminum plate, and was adjustable to the extent that it allowed postural orientation. Restraint straps provided standardized fixation of torso posture. The seat was constructed so that two primates could be exposed simultaneously to the acceleration environment. On the bottom of the vehicle was a 9 in. diameter, lead filled, thick wall steel cylinder onto which various diameter 1 in. steel cylindrical plates could be mechanically attached. The latter provided the means of programming the acceleration-time histories and served as the impacting head. The total payload weighed approximately 650 lb and was characterized by low center of gravity and excellent dynamic stability during both the free-fall acceleration and terminal impact

Aluminum honeycomb was found to be the most efficient energy absorbing brake. The hexagonal structure provides the unique property of complete dissipation of energy dur'ng constant load failure. The acceleration pattern was controlled by three variables:

1. Drop height.
2. Aluminum honeycomb specifications.
3. Total surface contact area of the impact head.

All impact time histories were recorded on high capacity (CEC) piezoelectric accelerometers mounted on the impacting head of the vehicle and beneath each primate seat. These accelerometers had a flat response of 2–6,000 Hz. The acceleration environments ranged 2–22 ms total time duration and 25–900 G peak acceleration. A baseline calibration of the electronic instrumentation was carried out before each experimental test drop.

Velocity measurements were accomplished by anchoring two photodiodes encased in an aluminum block on the impact vehicle. The diode faces were spaced 1 in. apart and all of the photodiode bodies were
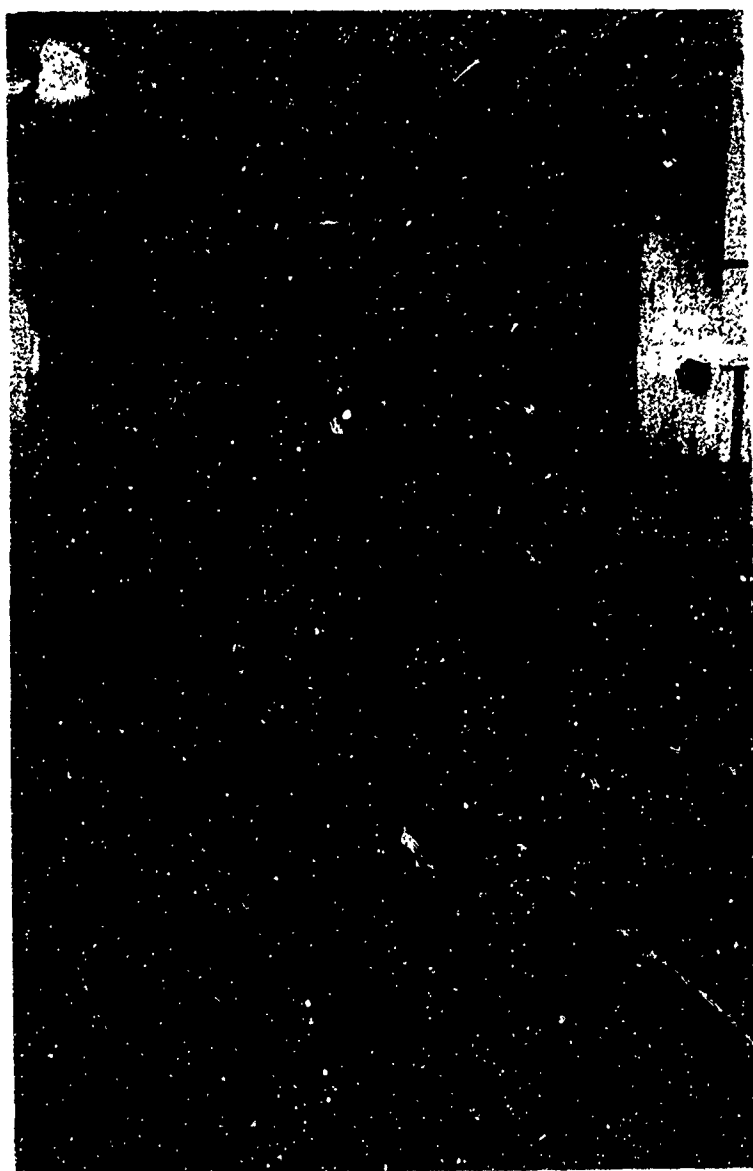
Fig. 1—Anesthetized primate supported and restrained on the impact vehicle

covered, with the exception of a 0.078 in. aperture over each face. A light source was magnetically coupled on the bulkward of the vertical decelera- tion tower assembly in line with the free-fall path of the photodiode aper- tures and 1 in. above the point of impact to provide for the velocity mea- surement. Each diode output was applied to a separate channel of the FM recorder. Terminal impact velocity was computed from the observed time differences between the two peaks of the photodiode's output signals, displayed on an oscilloscope, and measured by a Hewlett-Packard fre- quency counter during playback of the taped signals.

One hundred and four clinically screened Rhesus monkeys, ranging in weight 8–22 lb, were anesthetized with pentobarbital and surveyed radiographically to demonstrate normal bony vertebral spinal anatomy. If alteration in "normal" vertebral anatomy was present, the animal was rejected. Animals with a past history of acceleration exposure were un- acceptable.

Two experiments were conducted:

Experiment 1 — The upper limbs were suspended in such a manner that spinal flexion was minimized. The principal load bearing-load trans- mitting member was the vertebral column.

Experiment 2 — The upper limbs of the primate were tied together with nylon cord and attached to an "O" ring between the primate's thighs, exposing the vertebral body–intervertebral disk structure to asymmetrical load distribution.

These body positions were chosen primarily to study the effect of the alignment of the vertebral column on G_z tolerance and how these changes in position alter injury levels and mechanisms. (The two positions also attempted to reconstruct body positions and injury patterns occurring dur- ing ejection from disabled aircraft in order to provide practical guidance about the most desirable means of egress initiation.)

All primates were restrained in the vehicle in an upright position. The support and restraint system consisted of 1 in. nylon enforced cotton webbing and bite bar. A 1 in. lap strap was positioned across the bony pelvis. Sacrofemoral angle was 10–15 deg. A circumferential thoracic strap was placed around the chest approximately at the nipple line, an- chored to the seat back parallel with the seat pan, and tightened to a standard tension. A third strap restrained the distal lower limbs to pre- vent flailing. A bite bar was included primarily to immobilize the head and neck and minimize flailing motion.

The payload was raised to the experimentally predetermined height by a tower hoist assembly, released from rest by an electromechanical bomb shackle, and fell freely toward the impact programmer. At the moment of release, two Magniflux high-speed motion picture cameras (5000

frames/sec) were activated to record body dynamics during free fall and impact deceleration.

The acceleration environment used in this study resulted in a rectangular G-time history, which consisted of a short time-to-peak load in the microsecond range, a leveling off at essentially constant G level, and then a quick decay in the millisecond range. Fig. 2 is a representative acceleration-time history. The average acceleration was "eyeballed" and considered to be the level of the plateau. Since the rise time to the acceleration value was negligible to the pluse duration and essentially constant, the acceleration–time history was simply characterized by the plateau acceleration and total pulse duration.

Fig. 3 illustrates the resultant tolerance graph for the rectangular pulse distribution of the peak acceleration and pulse input along with the envelope of the vehicle and approximate data point scatter. The magnitude and duration of the rectangular acceleration pulses were chosen so that they covered the most probable range of injury predicted from pilot experiments and presently available scaling laws.

Immediately after impact exposure, the condition of each animal was ascertained by physical examination, including neurologic evaluation and anteroposterior (AP) and lateral spinal x-rays. Animals with roentgenographic vertebral changes, who were otherwise in good physical condition and without a detectable severe neurological deficit, were killed at variable intervals following impact exposure. The entire vertebral column was grossly dissected for morphologic study. Architectural derangements of trabecular patterns were compared to normal vertebral patterns macroscopically and by use of pre- and post-exposure radiographs. Muscle fibers and ligamentous and tendinous structures immediately adjacent to the vertebrae were not removed so that their participation in the energy dissipation mechanism could be observed. Primates that were paraplegic or otherwise severely injured were immediately killed and a detailed necropsy was performed with meticulous dissection of
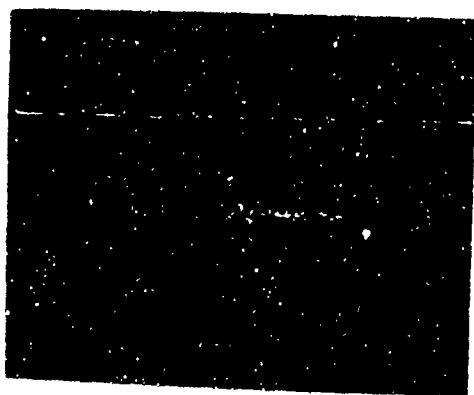
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Fig. 2   Representative acceleration-time history from a drop height of 120 ft, 120 G, 11 ms.

vertebrae, disks, spinal cord, and paraspinous muscles and ligaments. A complete necropsy was performed and representative tissue samples were prepared for histopathologic examination for each animal.

The acceleration-time history, high-speed photographic records of subject biodynamic response, and pre-, immediate, and post-roentgen-ographic changes, along with gross clinical and autopsy examination findings, were recorded, evaluated, and interpreted for all experimental exposures.

Injury to the internal organs was graded on an arbitrary scale incorporating the various degrees and intensities of trauma encountered for each organ system. For example, in the lungs massive peripheral hemorrhage accompanied by acute surface hemorrhage was considered a tolarance limit; for the liver, subcapsular hemorrhages, along with lacerations of the capsule and parenchyma, were considered to be an endpoint.

Although 11 distinct types of fracture patterns were observed, they were lumped into four categories:

1. Ten–15% loss of vertebral body height.
2. Anterior wedge fracture.
3. Comminutioı injury.
4. Spinous, transverse, and articular process fracture.

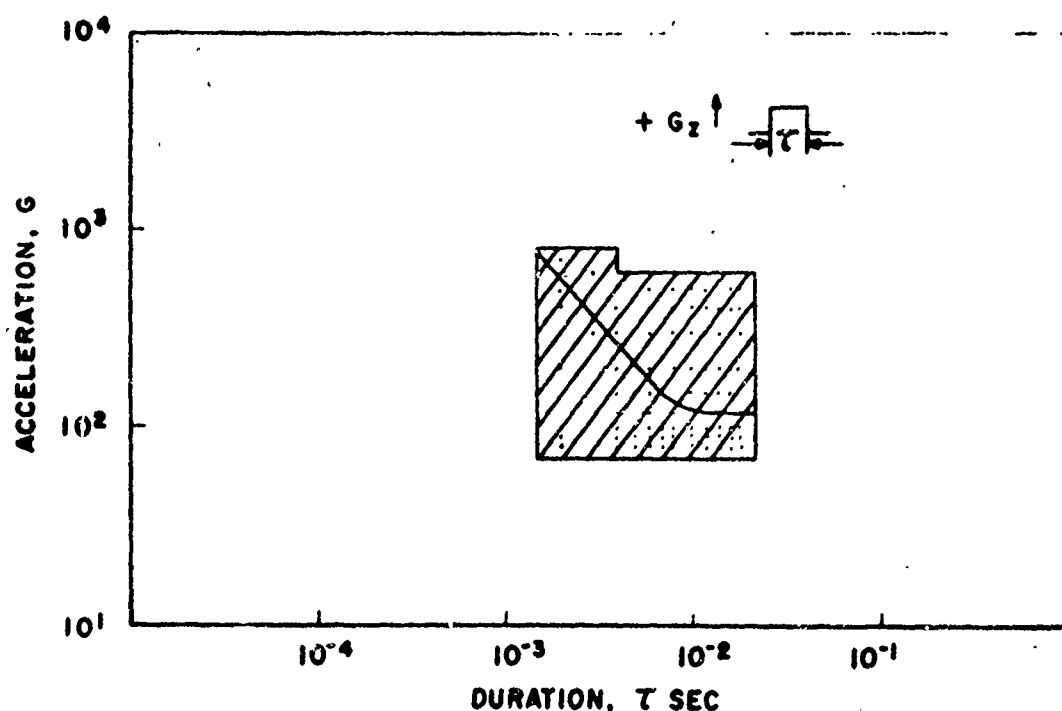Categories 1, 2, and 3 were used for the endpoint data presented.

Fig. 3—The approximate data scatter, envelope of the impact vehicle, and re sultant tolerance graph for the rectangular acceleration pulse. The magnitude and duration of the rectangular acceleration pulses were chosen so that they covered the desirable range of injury predicted from pilot studies and presently available interspecies scaling laws

Results

In these experiments, exposure to +G, longitudinal spinal impact resulted in lesions ranging from minor reversible abrasions to lethal injury. During necropsy, particular attention was directed to the spinal column and its surrounding structures. In all instances where fracture was described on post-impact exposure roentgenograms, it was confirmed by necropsy. In every instance where even minor fractures were present, the periosteum was usually torn and the anterior and posterior ligaments were either torn wholly or in part or were stripped from their points of attachment to the centrum of the vertebrae. Varying degrees of fraying and tearing of the interspinal and supraspinal ligaments in and surrounding the fracture site were accompanied by local hemorrhage from torn blood vessels. Extravasation of hematopoietic tissue into the surrounding soft tissue and bony marrow about the periosteum and endosteum and into the Haversian canals was observed.

Vertebral body fracture represented a primary mode of failure in the musculoskeletal system. Examination of both wet and dry bony specimens demonstrated two distinct types for each body position of structural bony failure: first, fractures involving loss of anterior vertebral body height, unilateral or bilateral dislocation, and narrowing of the anterior-posterior diameter of the spinal column; and second, uniform loss of vertebral body height with no significant associated vertebral body or articular facet displacement.

Experiment 1: Symmetrical Vertebral Body Loading—Uniform loss of vertebral body height and coarse grained cleavage fractures were observed when the translational bending of the thoracic spine was minimized. For these cases, few vertebrae were injured by short duration impact, with a decreasing number of vertebrae being involved as pulse duration decreased (G-level increased). Simultaneously, the primary fracture pattern changed from predominantly marginal plateau (fractures with uniform vertebral body crushing in effect by breaking down the fine trabecular of the vertebral body and by forcing the viscoelastic fatty tissue within the fine lamella and out of the perivertebral sinuses along with splintering of the surrounding thin cortical layer) to predominantly comminuted fractures (complete division of the vertebral body into several fragments with extensive splintering of the adjoining cortical bone).

In the AP view, radiographs revealed fracture patterns to involve a single vertebral centrum that was comminuted; the primary fracture path proceeded cephalically for approximately 4-8 mm and bifurcated. Each fork propagated at a small angle to the primary cleavage direction (roughly 30-50 deg.) In all cases the fracture pattern and direction of propagation was similar. The tail of the fracture path pointed downward.

In 22% of these cases, additional compression fractures were observed at considerable distances from the primary fracture site. Fig. 4 shows a sagittal section of the thoracic and lumbar spine with a comminution fracture at T_{11} and subluxation of L_3. Retropleural-retroperitoneal hematomas were observed in all cases.

Postdrop x-rays revealed a decrease in intervertebral disk space height, accompanied by an increase in convexity of the vertebral body end plates into the adjacent vertebral bodies in 68% of these primates. Necropsy demonstrated partial disruption of the anterior longitudinal ligament, subluxation of the posterior longitudinal ligament, injury to the cartilaginous epiphyseal plate, and subsequent loss in turgor of the intervertebral disk structure at the fracture site.

Injury to the articular facets occurred in association with comminuted fractures of the vertebral body. Injuries to the spinal cord by bone fragmentation or dislocation — or both — produced cord bruising or compression by the extravasation of blood or chronic alteration of the cord itself; in two cases, complete transverse lesions at the level of T_1 occurred. Tearing of the elastic interspinal ligaments at points of bony attachment was common. As the acceleration level was increased and total pulse duration was decreased, the lumen of the spinal canal was encroached upon by the displacement of bony fragments; the surrounding membranes were occasionally torn or ruptured, accompanied by the extravasation of blood, usually profuse, between the dura and the canal, amid the meninges. No evidence of thoracic cage fracture was noted in either experiment. Fractures occurred predominantly in the thoracic spine.

Fig. 4—Excised vertebral column of Macaca mulatta, sagittally sectioned, with a comminution fracture at T_{11} and subluxation of L_3

Experiment 2: Asymmetric Vertebral Body–Intervertebral Disk Load Distribution — Fractures involving loss of vertebral body height were observed most frequently in the thoracolumbar region. Postdrop exposure radiographs showed vertebral bodies approximated each other at the anterior and lateral periphery. Misalignment and loss of height at the anterior borders of the vertebral body in the region of the fracture site were usually accompanied by disparallelity or mechanical derangement — or both — of the articulating facets, irreversible deformation of the centrum, and increased dorsal kyphosis. The nucleus pulposus was driven dorsally toward the neural roots and capsuloligamentary sheath. The greater number of these fractures occurred at the level of the tenth thoracic and eleventh thoracic vertebrae. In the case of lateral vertebral body wedging (marginal plateau), a mild scoliosis was observed. Fig. 5 is a pre- and postlateral radiograph showing a minor compression fracture of T_{11} involving T_{10} and T_{12}.

Lesions of the spinal cord and its surrounding membranes were observed in 21% of these cases, and varied from simple compression to complete cord severance due to decrease in the anteroposterior diameter of the spinal canal. Edema of the spinal cord at the fracture site accompanied all of the above cases and was the most frequent cause of block in the subarachnoid space; it alone was sufficient to induce complete physiologic interruption in spinal cord function. As the acceleration level was increased and total pulse time duration decreased, the lumen of the spinal canal was encroached upon by the displacement of bony fragments; the surrounding membranes were occasionally torn or ruptured, accompanied by the extravasatian of blood — usually profuse — between the dura and the canal and amid the meninges. No dislocation of lumbar vertebrae was observed.

Fig. 5—Pre- and post-lateral spinal radiographs showing a minor compression fracture of T_{11} involving T_{10} and T_{12}. Retropleural-retroperitoneal hematomas were observed in all cases

Mild compression and forward fracture-dislocations of the cervical spine were usually accompanied by fracture of articulating facets and, in the more severe cases, fracture of the dorsal arch with forceful projection of bony fragments into the spinal canal.

Soft tissue injury, accompanying bone fractures in varying degrees depending on pulse duration and magnitude, was also observed but showed in general no difference for the positions of the two experiments. Therefore, the soft tissue results of the two experiments were combined.

The hemorrhagic lesions of $+G_z$ seated longitudinal spinal impact were found chiefly in five organ systems: lungs, liver, heart and great vessels, gastrointestinal tract, and central nervous system — in that order. Of these, pulmonary lesions were the most common. Injury result patterns to the CNS and gastrointestinal tract have not been completed and are not discussed.

The lungs and pleura are common sites of trauma at all acceleration-time histories. The severity of the lesions did not seem to differ in either experiment. In the lungs, signs of tissue damage ranged from edema and congestion to massive hemorrhage and laceration. Fig. 6 shows the general relationship of the severity of the lesions with respect to the acceleration-
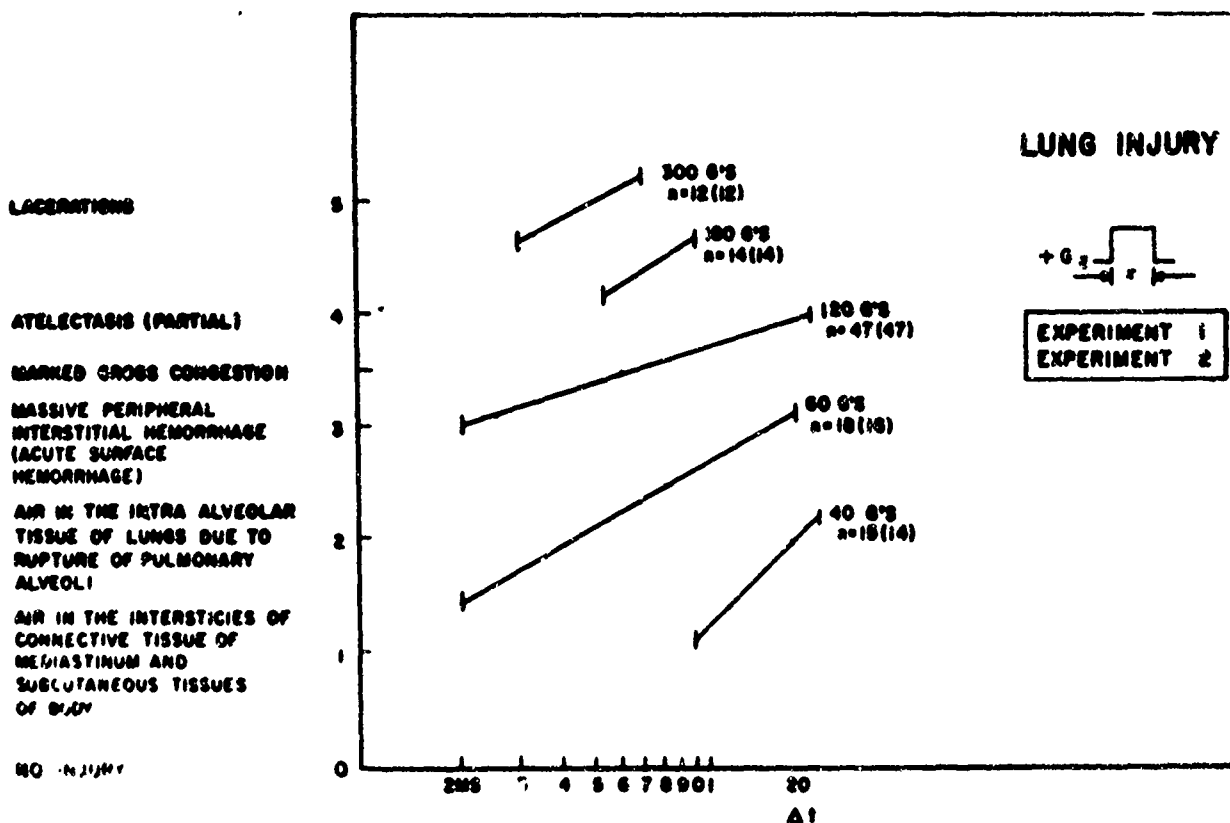

Fig. 6—The general relationship of the severity of pulmonary trauma to the acceleration-time histories. The n value is the number of animals exposed to the particular environment. The number inclosed by the parentheses is the number of animals receiving that particular type of trauma. Method of least squares was used to construct the straight lines

time history. Hemorrhage into the lungs was subpleural and distributed along the base of all the lobes at short acceleration-time histories. As the total time duration and G level increased, hemorrhage into the lungs was complicated by superficial irregular lacerations at the roots of the lungs, subpleural at the hila or at the base of the heart (or both) and resulted in a hemopneumothorax in 15% of all experiments. Contusion and laceration in the base of the lungs over the area in contact with the dome of the diaphragm extending 2-5 mm into the lung parenchyma were common. (The lungs of the Rhesus monkey differ from those of man with respect to an additional accessory lobe — lobus azygos — just caudal to the root of the lung, attached to the median surface of the inferior lobe, lying dorsally.)

Cardiac hemodynamics were not recorded throughout this study; however, spinal impact produced a bradycardia, most likely on a reflex basis, in all experiments. Postexposure radiographs revealed minor longitudinal axis rotation of the heart. Rupture of the aorta approximately 1 mm from the root was observed in five cases. In three of these five cases there was also tearing in the wall of the right auricle resulting in a hemopericardium. Primates exposed to a change in velocity greater than 48 ft sec commonly showed perforation of the pericardial sac and subendocardial petechia over the apex and base of the heart anteriorly, along with a marked hemorrhage into the soft tissue of the superior mediastinum, extending paravertebrally.

Myocardial contusions, lacerations, and rupture manifested by hemorrhage within the myocardium were relatively common findings for velocity levels greater than 40 ft/sec. Myocardial contusions of the right auricle were demonstrated by necropsy in 24% of all cases.

Another type of cardiac lesion observed during gross necropsy examination was the laceration of the leaflets of the valves along the bases of the tricuspid and mitral valves.

The viscoelastic plastic respiratory diaphragm is subject to high stresses resulting from the violent downward displacement followed by compression of the abdominal contents upward. The nature of the lesions ranged from hemorrhage into the musculature (which was occasionally observed), to tearing at the marginal points of attachment, and, in four cases, to extrusion of abdominal viscera into the thorax.

Lesions below the diaphragm were common. Numerous subcapsular hemorrhages of the liver measuring 0.5-2.0 cm were present in 89% of all cases. Subcapsular hematomas in the diaphragmatic surface of the liver and ragged lacerations lateral to the falciform ligament were observed. Occasional lacerations at the periphery of the superior convex surface of the right lobe involved the capsule and underlying hepatic parenchyma. Moderately large 1-3 cm areas of hemorrhage into the soft

tissue of the porta hepatitis were observed in all animals exposed to acceleration levels greater than 130 G. In 24% of these experiments, deep parenchyma lacerations radiating from the large hepatic veins near the terminations in the inferior vena cava were observed.

Ruptures of the intestines were accompanied by hemorrhages in the gastric mucosa in 29% of these cases. Multiple perforations of both small and large intestines were seen in 4% of the animals tested. These perforations usually accompanied lacerations of the mesentery.

The kidney is firmy anchored by a fibrosus network to the anterior surface of the posterior body wall. Traumatic lesions involving the kidney included interstitial hemorrhage and perirenal hemorrhage and were not considered significant.

The spleen and pancreas were not a common site of traumatic lesions; however, laceration of the spleen occurred in 6% of these cases. Injury to the pancreas was encountered but was limited.

Gross necropsy of the remaining organs was performed but without significant findings.

Fig. 7 illustrates the 99% probability of injury for the Rhesus monkey exposed to a square wave input and $+G_z$ acceleration vector. It also
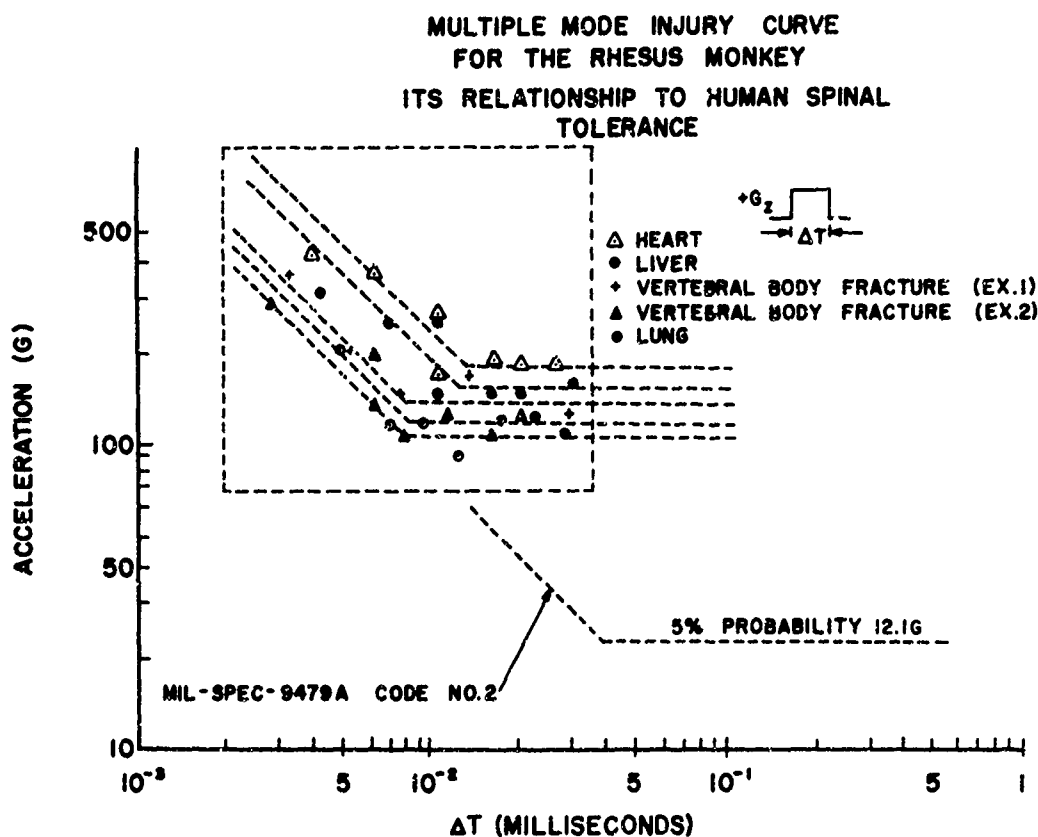

Fig. 7—Illustration of the multiple mode injury for the 99% probability of injury for Rhesus monkey. The fundamental mode of injury is lung damage, followed by vertebral body compression, liver damage, and injury to the heart. The rectangle inclosing the injury curves is the envelope. of the vehicle. The critical impact velocity is 41 ft/sec. Superimposed onto this curve is the 5% probability of injury for 1 G, human exposure with a rectangular acceleration pulse (from Military Specification 947/9A)

shows several randomly selected points for each mode of injury. The fundamental mode of injury is trauma to the lungs, followed by vertebral body compression and contusion of the liver. The data scatter indicated a linear regression existed for each injury mode. The method of least squares was used and was found to provide a reasonable fit. The corner duration was approximated. The dashed lines are the theoretically predicted slopes for each injury mode. Superimposed onto this graph is the 5% probability of injury for man. For very short durations (the duration of the pulse being less than the natural period of the system for the particular body position) the change in velocity represented by the acceleration pulse is the only significant parameter. For the longer duration periods, or when the pulse duration is greater than the natural period of the system, the peak acceleration is the biologically significant factor. These results confirmed the earlier experimental findings of Kornhauser and Lombard, borne out by the theoretical prediction of Payne, Kornhauser, and others.

Discussion

The response of the primate torso to impact excitation is primarily mechanical in nature. When the primate system is exposed to seated abrupt acceleration, the pelvic structure and vertebral column act as the prime load-bearing, load-transmitting elements. The polyphasic parenchymatous and hollow structures contained within the abdominal, thoracic, and cranial cavities do not respond to the applied loading as a unit mass; rather, impulsive mechanical excitation has been shown to trigger responses of many modes (longitudinal, torsional, transverse) at a series of frequencies dependent upon the character of the particular forcing function.

The degree of excitation of an individual organ is a function of its location, mass, physical dimensions, elastic limits of suspensory, and accessory attachments along with the point and method of attachment to the surrounding structures.

At certain critical frequencies, the relatively unsupported organs within the pelvic and abdominal cavity were observed to move as a semi-viscoelastic mass and oscillate freely within the confines of the abdominal cavity. These hydrodynamic characteristics were observed on 16 mm motion picture film and delineated as follows:

1. **Free-Fall Stage** — The organs within the abdominal cavity respond to the free fall in their individual manner, but in general are forced upward against the dome-shaped musculofibrosus respiratory diaphragm, stretching the intestines and mesentery. At the instant of impact, the pelvis, sacrum, and vertebral column act as the prime load-bearing, load-transmitting structures surrounded anteriorly by tissue onto which the

inelastic (rate dependent irreversibility and rate dependent effects) anisotropic organs are anchored. These parenchymous and hollow structures within the cavity of the torso, unable to respond immediately to the impact deceleration, exhibit a complex mechanical behavior.

2. Impact Stage — Shortly following impact, the abdominal contents are forced downward (the volume of the gas in the lungs is considerably increased); a time varying mechanical and volumetric distortion occurs at the level of diaphragm and thoracic chest. The entire torso commences to compress under decelerative loading. As it arrives at a level of maximum compressive deformation, a fullness develops under the surface of the skin (Fig. 8) in the region of the lower pelvis, expanding radially outward and propagating cephalically as a spherical wave front. This surface wave is propagated parallel to the vertebral column, and in opposition to the inertial response of the torso. (An elemental area of the surface perpendicular to the torso executed an elliptical path.)

Fig. 8—Abdominal surface wave propagation as a result of $+G_z$ rectangular acceleration impulse. The surface wave is propagated parallel to the vertebral column and in opposition to the inertial response of the torso.

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3. Expansion Stage — As the surface wave travels up the torso, its amplitude increases and its velocity decreases, indicating a change in the associated stress magnitude as the wave propagates. The surface wave engulfs the entire abdominal torso and approaches the thoracolumbar diaphragm (the muscular partition separating the abdominal viscera and inclosing peritoneum from the pleura and pericardium). At the level of the diaphragm, the characteristics of the wavefront are transformed, due to a complex interaction occurring between the advancing surface wave front and the viscoelastic diaphragm. The transmission of the disturbance takes about 6 ms to transverse the abdominal torso.

The diaphragm and thorax act both as a reflecting and an attenuating barrier. As a result, the energy content of the wave is split up into several externally observable modes.

Initially, wave reflections occur and cause unloading waves to be propagated down the abdominal torso that subsequently undergo reflection at the area of the pelvic diaphragm and travel cephalically.

As the change of velocity and acceleration-time history is increased, the surface wave, in addition to being reflected, interacts with the on-coming upper torso mass. Consequently, whole body compressive deformation is abruptly terminated and reverses direction, the surface wave transverses into the thoracic chest to be attenuated by the restraint across the chest wall, occurring approximately at the nipple line.

4. Final Stage — During this period, the forces continue to act in a direction opposite to the primary deceleration force, and result in a gradual transformation of kinetic energy of motion of the displaced organs into elastic energy of the supporting skeletal mass. The final form of this elastic energy is limited only by the viscoelastic-plastic properties of the various body tissues. The torso mass, the thoracic mass, and the diaphragm return to the pre-impact position after their entire energy content is dissipated.

The character of the observed surface disturbance is assumed to be a result of the violent displacement and deformation of the intraabdominal organs. The state of tissue deformation in the abdominal torso varies both with respect to time and position.

One visible critical boundary is the respiratory diaphragm which acts as an elastic barrier for the accelerating abdominal mass and probably severely distorts the topographic-anatomic interrelationship of the thoracic viscera.

So long as the stress-strain-time relationship produced deformations within the elastic limit of the organ system, the internal mechanical energy created was absorbed and dissipated by the individual cells, tissue, and fluidic transport system. However, when the stress-strain-time relationship exceeded the elastic limit of a particular organ due to excessive mechanical

loading, nonhomogenous deformations involving rearrangement of tissue resulted. Thus, the continuity of energy transmission was interrupted, initiating complex viscous and plastic tissue disruption.

The degree of energy transmittal, near the free surface of the skin to the various internal tissues and organs, remains open for discussion. Although it can be speculated that the vertebral column acts as a transmission corridor for stress propagation, the answers to these questions remain unknown until suitable methodology is developed to quantitate these observations in the living animal.

A variety of animals have been used in acceleration experiments including Rhesus monkeys (36), bears (37), chimpanzees (38), mice (39), and guinea pigs (40). Kornhauser's experiments (41) with mice showed reasonable confirmation of the tolerance curve predicted by a simple dynamic model, and concluded that a biological systems response to acceleration was equivalent to the output of a mechanical system. Theoretical consideration indicates that geometrically similar structures should have similar tolerance curve shapes with a sensitivity and frequency shift that remains a function of the particular species under test.

Experiment 1: Symmetrical Vertebral Body Loading — The hard tissue disruption observed in this experiment was rather straightforward. At low G levels, post-impact radiographs showed that vertebral bodies approximated each other at the periphery, along with occasional irregular contours of the adjacent cartilaginous end plates. The fractured vertebrae were "uniformly flattened," a type of injury that is easily overlooked.

As the change in velocity was increased and total time duration decreased, post-impact radiographs showed fracture patterns to be invariably "Y" type with well defined cleavage planes. Macroscopic and gross examination of wet and dry bony specimens indicate the trabecular bone of the vertebral body behaves purely elastically, and fractures at a given strength without permanent strain, suggesting an increase of the yield and ultimate strength of the vertebral bone with increasing rate of strain.

These observed differences in fracture type indicate that there may exist a critical transitory period for spinal segments, such that at certain velocity-time histories, the energy within the encapsulated incompressible nucleus pulposus and annulus fibrosus of the intervertebral disk is homogeneously distributed over the internal surfaces of the contiguous cartilaginous end plates. The viscous marrow exhibits a time-dependent mechanical behavior as it is displaced through the numerous vascular channels of the vertebral body. However, when the energy content of the acceleration pulse is increased, the vertebral body fractures by the mode that requires the least amount of strain.

Examination of the cartilaginous end plate indicates the incompressible

nucleus pulposus may act as a stress concentrator and consequently a fracture initiator.

Experiment 2: Asymmetrical Vertebral Body Loading — The downward displacement of the abdominal viscera allowed several qualitative observations upon later review of the motion picture film. In all cases there occurred a pronounced extension-flexion of the cervical spine, due to the rotary inertial characteristics of the head, along with acute flexion of the thoracic spine that decreased the weight-bearing surface areas of the spinal motor unit (intervertebral disk complex) and adjacent bony structure by a factor of approximately three (42).

The trauma associated with this body position was extensive, complex, and difficult to isolate, in terms of playing havoc with the vertebral column and associated musculature.

Transverse sections of excised intervertebral disks adjacent to primary fracture sites exhibited minute fissures in the annulus fibrosus extending posteriorly (toward the spinal canal). This syndrome may be a sequal to traumatic posterior protrusion of the annulus fibrosus without herniation of the nucleus pulposus or posterior intervertebral disk prolapse (or both), hypertrophy of the ligamentous flavum, and nerve root irritation or compression. These results will be given in a separate paper yet to be published.

The pathologic lesions produced experimentally in the Rhesus monkey are similar in their location and nature when compared to human clinical case histories, and varied only in their incidence and severity. The mechanisms by which these lesions were produced seem to be similar. As factors such as skeletal mass, body configuration, and whole body center of gravity become available, precise dynamic and physiological interspecies scaling criteria will be established for the assessment of human acceleration soft and hard tissue tolerance limitations.

However, much work is still necessary in order to identify the modes of injury to the various organs and their subsystems, and to further validate and apply these results as a tool to assess the human injury spectrum and minimize trauma; nevertheless, the results are encouraging. One major problem is that the present knowledge relative to the dynamic behavior of a biologic tissue exposed to abrupt acceleration is still in its infancy. The dynamic mechanical properties of the rate-sensitive body organs have not been adequately considered in the design of support and restraint hardware. Data concerning optimal load analysis for individual body segments, their characteristic load-bearing areas, and the identification of load paths within the torso for particular acceleration-time histories is sparse. Likewise, in order to establish dependable analytical and hardware models, the mechanical properties of soft tissue, the musculo

skeletal response — along with the nonlinear viscoelastic-plastic response — of individual organs and their subsystems and physiologic function must be considered.

Summary and Conclusions

The aim of this work , s to collect experimental data to describe the biodynamic response of the Macaca mulatta to $+G_z$ spinal impact, so as to establish more precisely the dynamic, anatomic, and physiologic scaling criteria necessary to provide a rational basis for extrapolating animal impact data to man.

The curves of equal injury severity (tolerance curves) obtained exhibit— as a function of acceleration magnitude and duration—the behavior and the general sensitivity expected from theoretical prediction. The shape of the curves obtained for soft tissue injury supports the hypothesis that to a first approximation, abdominal viscera and diaphragm are deformed and stressed in a conphase motion without individual pronounced organ resonances. The lungs were, in general, found to be most sensitive to trauma, with liver and heart injury following in that sequence. Differences in the alignment of the spinal column during the impact exposure did not markedly alter soft tissue damage levels.

Vertebral body and intervertebral disk injury were, however, critically dependent on the symmetry of spinal loading. Symmetrical loading not only resulted in approximately 20% higher loads for equal injury severity, but also produced completely different fracture patterns. Whereas symmetrical loading tends to produce fractures confined to two or three vertebrae, asymmetrical loading immediately involves five to seven vertebrae, depending on acceleration magnitude and duration.

The results of the study explain and emphasize the difficulties encountered in defining with more precision curves of equal injury probability or severity because of the differences in injury mechanisms for different time functions of the loading force. These difficulties already exist for the mechanical trauma alone — under the best controlled exposure conditions — without reference to the large spread of the physiological and performance capability consequences of the trauma. In addition, cardiac hemodynamics and respiratory phase at the instant of impact are important factors: they play a role in the mechanism and degree of injury and should be monitored.

Acknowledgments

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